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NAS REPORT ON TECHNOLOGICAL FEASIBILITY OF 1975-76 MOTOR VEHICLE EMISSION STANDARDS

A CRITIQUE OF THE 1975 FEDERAL AUTOMOBILE EMISSION STANDARD FOR CARBON MONOXIDE, NATIONAL ACADEMY OF SCIENCES

NATIONAL ACADEMY OF SCIENCES

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This report covers the results of a study, by the Panel on Emissions Standards, Committee on Motor Vehicle Emissions, NAS, into the basis for the Federal motor vehicle carbon monoxide emission standard for 1975 and later model year vehicles. The study covers the measurement of ambient CO levels, health effects, growth factors, sources, air quality models and computation of emission standards.

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Errata

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National Academy of Sciences

May 22, 1973

Page 8, Figure 1, caption: Line 8 should read:

$(0.01 < p < 0.02$ at 50 ppm; $0.001 < p < 0.01$ at higher doses.)[2]

Page 11, Figure 3, caption: Change "McIlvaine *et al.* (7)" to "Smith, R. G. (7)"

Page 38, Reference 7; Change to:

7. Smith, R. G., Discussion of paper by B. D. Dinman: "Pathophysiologic Determinants of Community Air Quality Standards for Carbon Monoxide," J. Occupational Medicine, vol. 10, p. 461, September, 1968.

Table A-1 (following page 41); in column headed "References", change reference numbers as follows:

57 to A-1; 81 to A-2; 82 to A-3; 59 to A-4; 56 to A-5
58 to A-6; 86 to A-7; 124 to A-8; 83 to A-9; 61 to A-10;
62 to A-11

A CRITIQUE OF THE 1975 FEDERAL AUTOMOBILE EMISSION STANDARD FOR CARBON MONOXIDE

Prepared by the Panel on Emission Standards

for

COMMITTEE ON MOTOR VEHICLE EMISSIONS

NATIONAL ACADEMY OF SCIENCES

May 22, 1973

NOTICE

The Committee on Motor Vehicle Emissions has evaluated the technological feasibility of meeting the light-duty motor vehicle emissions standards as prescribed by the Clean Air Amendments of 1970. This study was performed under the sponsorship of the National Academy of Sciences and with the express approval of the Governing Board of the National Research Council.

The Committee obtained much of its information from eight panels of consultants, each panel dealing with a particular subject area of importance in the Committee deliberations. Panel members were selected by the Committee on the basis of recognized competence in specific areas.

The panel reports are reports of the panels to the Committee. Before publication, each panel report was reviewed by appointed members of the Committee. The views represented by the panels are one of the sources of information provided to the Committee and were used as a partial basis for the Committee judgments.

PREFACE

In legislating the Clean Air Amendments of 1970, the Congress asked the Environmental Protection Agency to contract with the National Academy of Sciences to conduct a comprehensive study and investigation of the technological feasibility of meeting the motor vehicle emissions standards prescribed in accordance with the law. In responding to this request, pursuant to a contract with the Environmental Protection Agency, the Academy established a Committee on Motor Vehicle Emissions and charged it with the conduct of this study. The Committee published the results of its work in a report to the Environmental Protection Agency dated February 12, 1973.

As a means of providing itself with authoritative information and expertise in the various critical aspects of the problem it undertook to study, the Committee appointed specialist panels to undertake investigations and report their findings. The following report, A Critique of the 1975 Federal Automobile Emission Standard for Carbon Monoxide, presents the findings of one of those panels.

Taken together, the special panel reports, of which this is one, constitute a very substantial accumulation of data and analysis brought together by many specialists in many investigations in a very fast-moving area of technological development. In its published report, the Committee on Motor Vehicle Emissions has, of course, brought together that part of all this information and analysis required to fulfill its stated obligation to the Environmental Protection Agency. The separate reports of the specialist panels are published for the public record and to complete the documentary record.

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The Panel acknowledges the efforts of its Technical Assistant, Richard Kamens, in the preparation of this report. He in turn, was assisted by Drs. Guiseppe Fumarola, University of Genoa, Italy, Yuji Horie and John Overton of the EPA-supported research project #1 R01 AP01130-01 "Critique of Air Quality Management Standards" at the University of North Carolina at Chapel Hill.

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1. Carbon Monoxide

The most recent comprehensive report on carbon monoxide is "Air Quality Criteria for Carbon Monoxide -- A Report by the Expert Panel on Air Quality Criteria," Committee on Challenges of Modern Society, North Atlantic Treaty Organization [1]. It consists of eight chapters, each of which concludes with a summary. The summaries of chapters 1, 2, 3, 4, and 6 are reproduced in the material that follows in this chapter and provide an up-to-date statement of the status of subject matter not otherwise discussed in this report.

Occurrence, Properties, and Fate of Atmospheric Carbon Monoxide

Carbon monoxide, which constitutes the largest single fraction of the pollutants found in urban atmospheres, is produced primarily by the incomplete combustion of organic materials used as fuels for both transportation and space heating; it also results from industrial processes, refuse burning, and agricultural burning.

Although several natural sources of CO of both biological and nonbiological origin have been identified, their influence on the CO concentration in air in cities is considered to be negligible. The CO background concentration of clean, uncontaminated air has been found to be 0.15 to 0.16 mg/m^3 (0.13 to 0.14 ppm) in the northern hemisphere and 0.06 mg/m^3 (0.05 ppm) in the southern hemisphere.

Carbon monoxide is colorless, tasteless, and odorless; although flammable, it does not support combustion. Oxidation reactions that convert CO to CO_2 occur with both oxygen and water vapor in the lower atmosphere, but the rates of these reactions are very slow.

Worldwide emissions of CO from technological sources have been estimated to be more than 1.8×10^{11} kg (200 million tons) annually. In the absence of any removal processes, this large tonnage would be sufficient to raise the background level of CO by 0.03 mg/m^3 (0.03 ppm) per year; yet this background level does not appear to be rising. The mean residence time of atmospheric CO has been estimated to be between 0.2 and 3 years.

Several removal processes are known that explain the constancy of the CO background. The oxidation of CO by OH radicals seems to be the most important sink. Additionally, CO is removed by terrestrial biospheres, through processes such as the metabolic conversion of CO to CO_2 and methane by soil microorganisms; the binding of CO to porphyrin compounds in plants and animals, with subsequent oxidation of the CO to CO_2; and the adsorption and subsequent oxidation of CO on various surfaces. Although the biosphere provides several potential mechanisms involving both land and marine plant and animal communities, the precise mechanism of removal of CO from the atmosphere cannot at present be identified with any certainty. In fact, some of the CO sinks discussed above are highly speculative and may actually contribute CO to the atmosphere, rather than remove it.

Principles of Formation and Control of Carbon Monoxide

Carbon monoxide is formed predominantly by incomplete or inefficient combustion. If any of the four variables that control this process are low, CO emissions may be high. These variables are oxygen concentration, flame temperature, gas residence time at high temperature, and combustion-chamber turbulence. In the internal combustion engine, the air/fuel ratio controls the CO concentration in the exhaust, whereas the total volume of exhaust relates to power output. In stationary sources, combustor design and operation determine the CO emissions.

Estimation of Carbon Monoxide Emissions

Annual carbon monoxide emissions from major sources in the United States were estimated to be 137×10^9 kg (151 million tons) for the year 1969. In the Federal Republic of Germany, annual CO emissions for 1969 were estimated to be 12.8×10^9 kg (14 million tons). In the United States, the largest single source was the gasoline-powered motor vehicle, which accounted for 88×10^9 kg (99 million tons), or over 64 percent of the total. Internal combustion engines used in aircraft, non-highway use, trains, ships, and diesel-powered motor vehicles accounted for another 10 percent of the total. Other major sources included fuel combustion in stationary sources, industrial processes, solid-waste combustion, and forest fires.

Although carbon monoxide emission totals were found to differ among different regions of the United States, the highest estimates ranged up to about 5×10^9 kg (5 million tons) per year in New York and Los Angeles. In all but 2 of 26 cities surveyed, the CO emissions were the largest component of the total tonnage of pollutants emitted to the atmosphere. The major source of CO emissions in all of the regions studied was the motor vehicle.

Generally, it can be said that in the concentrated-traffic areas, the CO emissions from motor cars will predominate. In order to get detailed information, however, it will be necessary to make intensive investigations regarding the local and temporal distribution of CO emissions from all emitters -- traffic, industries, domestic furnaces, etc.

Measurement of Carbon Monoxide Concentrations in Ambient Air

Because CO levels exist over such a wide concentration range, the use of a wide variety of chemical and physical procedures may be required to adequately evaluate atmospheric concentrations. The accuracy of any method used can be no better than the standards used to calibrate the instrument or method used. When pure CO and CO-free nitrogen are available, volumetric gas dilution techniques are satisfactory ways to prepare standard gases. Gas mixtures of doubtful or unknown concentrations of CO may be assayed by the gravimetric and chemical (iodine pentoxide) standardization methods. These procedures are time-consuming, but accurate.

The continuous measurement of atmospheric CO can be accomplished by using a variety of techniques. The most commonly used device is the nondispersive infrared (NDIR) analyzer, which is sensitive but is subject to a number of interferences. These interferences, however, may be removed or minimized by proper treatment of the air stream.

New NDIR analyzers can measure CO in the range of from 1 to 29 mg/m^3 (1 to 25 ppm), and can be equipped with automatic zeroing, spanning, and calibrating equipment. In addition, they have the advantage of being linear in output. NDIR systems have the advantages of being insensitive to flow rates, of requiring no wet chemicals, and of being reasonably independent of ambient air temperature changes if the instrument is properly thermostated. The NDIR analyzer is generally accepted as being the most reliable reference method for the calibration of other instruments.

Continuous CO monitoring instruments based on galvanic and coulometric principles have not been used extensively in field operations. They are flow- and temperature-dependent and require the use of filters to remove multiple interferences.

The mercury vapor analyzer, which depends on the liberation of mercury vapor when CO is passed over hot mercuric oxide, has been used as a portable, continuous monitoring analyzer with a special capability for measuring low CO concentrations (0.29 mg/m^3 or 0.25 ppm). The instrument does not appear, however, to be suitable for routine CO monitoring of urban atmospheres.

The automated gas chromatographic system is semicontinuous in operation but has the advantage of measuring both CO and methane. Concentrations of from 0.1 to 1150 mg/m^3 (0.1 to 1000 ppm) may be determined, and instrument output for both gases is linear over the range mentioned. Instruments of this kind may be available soon, and it appears this technique will be useful for measuring CO in heavily polluted areas as well as at remote, relatively clean locations.

CO monitoring systems based on catalytic conversion principles are used widely in enclosed spaces, but their applicability for ambient air monitoring is limited.

Colorimetric (colored silver sols) methods are not recommended for monitoring ambient air. The approach may be useful, however, for measuring CO in stack effluents. Colorimetric techniques and length-of-stain discoloration methods are recommended for use only when the above-mentioned physicochemical systems are not available. They can be used in the field where accuracy is not required and can be of great value during air pollution emergencies.

Effects of Carbon Monoxide on Plants and Certain Microorganisms

Carbon monoxide has not been shown to produce detrimental effects on higher-type plant life at concentrations below 115 mg/m^3 (100 ppm) during exposures of 1 to 3 weeks. Nitrogen fixation by free-living bacteria was inhibited at exposures of 2,300 mg/m^3 (2,000 ppm) CO for 35 hours. Nitrogen fixation by efficient nitrogen-fixing bacteria in clover roots was also inhibited by 115 mg/m^3 (100 ppm) CO when exposed for 1 month.

Ambient CO levels rarely reach 115 mg/m^3 (100 ppm) even for very short periods of time. In view of this and the foregoing information concerning CO effects, a significant impact on vegetation and associated microorganisms seems improbable.

It should be pointed out that no information is available on CO concentrations in soils. Most higher plants grow with their roots in the soil, and nitrogen-fixing organisms as soil dwellers would be vulnerable to soil-borne CO. Hundreds of species of both flora and fauna, whose activities are important to soil development and to plant growth, may also be affected.

2. Health Effects of Carbon Monoxide

Carbon monoxide combines with the hemoglobin of the blood in almost the same manner as oxygen, except that the affinity of hemoglobin for CO is almost 200 times greater than for oxygen. The carboxyhemoglobin, COHb, formed from the reaction of CO with blood hemoglobin, reduces the quantity of hemoglobin available for oxygen transport. The presence of COHb also reduces the ability of oxygenated hemoglobin, O_2Hb, to release oxygen to the tissues. The level of COHb in the bloodstream is expressed as percent saturation, the portion of the total available hemoglobin tied up as COHb. Intrinsic metabolic processes produce a "background level" in a normal person of about 0.5 percent COHb. COHb levels higher than 0.5 percent in normal people may be attributed to the CO in the air respired over a given time. Thus, it is of concern to understand the relationship between CO concentration and COHb levels and the health effects associated with various levels of COHb.

No evidence has been presented to indicate that there are significant health effects when COHb is below 2 percent. There is evidence that persons with pulmonary or cardiovascular disease are at risk when COHb is above 5 percent. There is lack of agreement as to whether or not the performance of tasks requiring time, sight, or sound discrimination is affected by COHb levels between 2 percent and 5 percent. There is even question as to whether task performance of persons without pulmonary or cardiovascular disease suffers below 10 percent COHb.

Experiments on Task Performance When COHb is between 2 Percent and 5 Percent

In 1967 Beard and Wertheim [2] reported a study relating possible impairment of task performance because of carbon monoxide exposure. Eighteen university students were indivdually exposed to CO concentrations ranging from 0 to 250 ppm. During the exposure, the subjects were asked to compare the duration of a tone to a standard signal which lasted for exactly one second. The subjects did not know if the signal was longer, shorter, or the same length as the standard signal and they were given no intentional indication of the correctness of their response. Beard and Wertheim used a single-blind test

protocol, i.e., the subject, but not the experimenter, was unaware of the CO concentration being administered. Some of the results from this study are given in Figure 1. COHb was not measured, but COHb concentration in blood can be estimated from Figure 2[3]. From Figure 2, it can be seen that the 50 ppm point on Figure 1 is equivalent at 90-minute exposure to 2.5 percent COHb. Thus the Beard and Wertheim data indicate an impairment of task performance at this COHb level.

Stewart et al.[4] performed a similar experiment, but they used a double-blind test protocol, i.e., both the subject and the person administering the test were unaware of the CO concentration being administered at any given time. The subjects in the Stewart et al. experiments were exposed as a group and the performance tests they took were different from the ones given by Beard and Wertheim. Stewart et al. maintained the CO concentration more closely than Beard and Wertheim (Beard and Wertheim, ± 10 percent; Stewart et al., ± 2 percent) and measured COHb during the experiment. Stewart et al. were unable to observe any impairment of task performance at exposures of 100 ppm for 8 hours (11 - 13 percent COHb).

It is clear that the Beard and Wertheim experiments must be confirmed. However, if their results are correct, it must still be demonstrated that the type of impairment that was observed at 2.5 percent COHb is functionally significant.

Patients with Cardiac and Pulmonary Impairment

DuBois[5] states that it is reasonably certain that 8 percent COHb is too much for patients with angina pectoris or myocardial infarction. There appears to be a general consensus that a maximum of 5 percent COHb should not be exceeded in persons with cardiac or pulmonary difficulties.

Carboxyhemoglobin Levels and Ambient Carbon Monoxide Concentrations

Individuals are not usually exposed continuously to the very highest urban carbon monoxide ambient concentrations for long periods of time. A person may be in freeway traffic for one hour, walk downtown through a busy intersection, and then spend a good part of the day in a lower carbon monoxide environment. However, there are people who spend

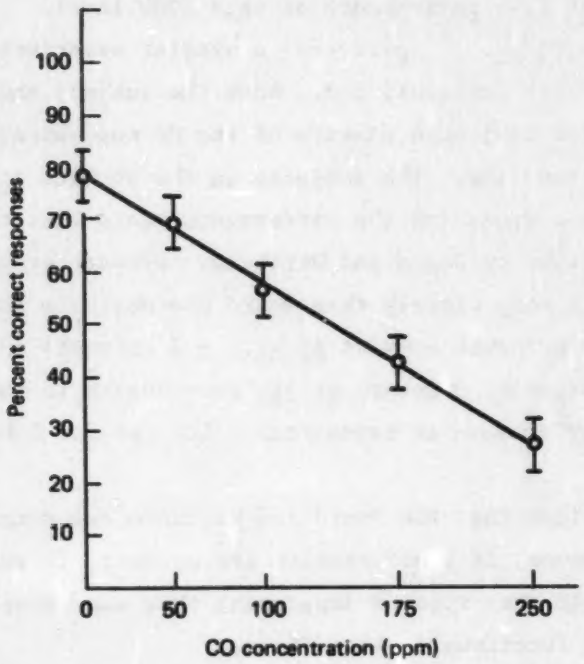


FIGURE 1 Mean percent correct responses, ±1 s.d., by 18 human subjects, during the discrimination of two tones presented successively. Each point is the mean of three determinations per subject, at each CO dose, of performance during the second and third hours of the session, i.e., during 0.5-2.5 hours of CO exposure, since exposure began after the first 0.5 hour. Comparison of performance in sessions during and prior to CO revealed significant impairment (t-test) at all doses ($0.01 < p < 0.02$, at 50 ppm; $0.001 < p < 0.01$ at higher doses.[2]

Source: Beard, R. R., and Werthein, G. A., Behavioral impairment associated with small doses of carbon monoxide, Amer. J. Publ. Hlth., vol. 57, p. 2011, 1967.

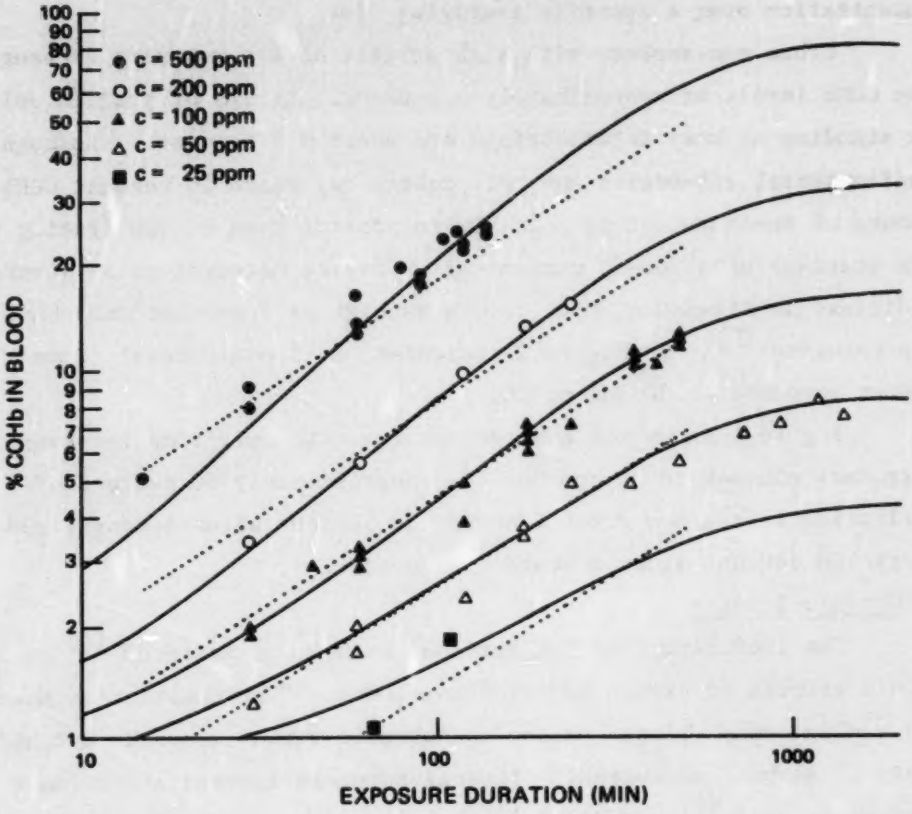


FIGURE 2 Data relating blood COHb with exposure to various concentrations of carbon monoxide. A regression equation (dashed lines) and a more complex equation by Coburn (3) (solid lines) were fitted by Peterson and Stewart to the data points in Figure 2. The Coburn equation provides a better fit.

considerable time during the day exposed to emissions from high-density traffic. Street vendors and newspaper streetside concessionaires are a few examples. Because of the difficulty of monitoring an individual's CO exposure, there seems to be agreement that his COHb level is a better measure of his CO exposure than are average values of ambient CO concentration over a specific averaging time.

Urban non-smokers with high ambient carbon monoxide exposure have COHb levels of approximately 2 percent. Levels of traffic policemen standing at busy intersections are about 2.5 percent. Although traffic tunnel attendants and toll takers may reach 10 percent COHb after 8 hours of exposure, it is possible to protect them by ventilating their work stations with low-CO content air. During meteorological inversion conditions in Milwaukee, COHb levels as high as 4 percent COHb have been recorded [6]. As Figure 2 indicates, this could result from 8 hours exposure to 30 ppm of CO.

Figure 3 shows the average percent COHb over time in seven nonsmokers exposed to 30 ppm CO [7]. Approximately 60 percent of equilibrium is reached after 2 hours; 80 percent after 4 hours; and almost 100 percent after 8 hours of exposure.

Chronic Effects

The 1969 report of The National Academy of Sciences [8] on chronic effects of carbon monoxide concludes: "The question of whether such a clinicophysiologic entity as 'chronic carbon monoxide intoxication' exists is as yet unanswered. Several European investigators (most prominently Grut [9] and Petri [10]) believe that such a condition does exist, but the consensus in the United States is that the syndrome has not been adequately defined."

If an exposure to a high concentration of CO results in sufficient oxygen deficiency to cause a pathological change, this change may not be reversible. This is an acute, rather than a chronic manifestation, and there is no disagreement about its occurrence. The issue is whether cyclic exposure to low levels of CO, such as might produce 5 percent COHb, and to background levels during which COHb will drop to the 0.5 percent level, will cause a disease entity in

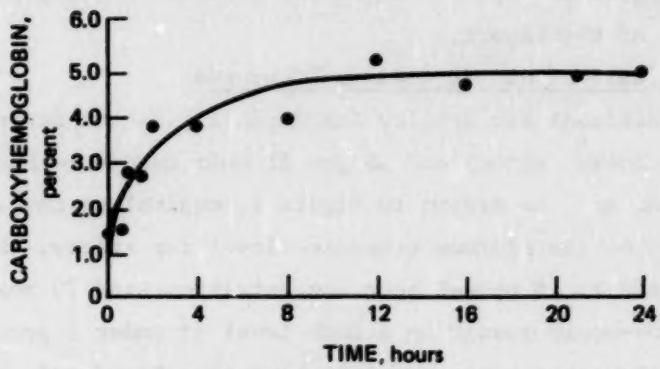


FIGURE 3 Average values of percent carboxyhemoglobin in seven non-smokers exposed to 35 mg/m^3 (30 ppm) CO. Source: McIlvaine et al. (7).

otherwise health people. As noted above, most workers in the field of CO effects, both in Europe and America, do not believe that there is such a disease entity. In other words, it is believed that there is no permanent or chronic effect caused by the changes in COHb that occur daily in any individual exposed to low level concentrations of carbon monoxide.

Summary of Carbon Monoxide Toxicology and Epidemiology

Chapters 7 and 8 of the NATO report "Air Quality Criteria for Carbon Monoxide" [1] discuss the health effects of carbon monoxide. The summaries of these chapters are reproduced in this report in Appendix A, although they repeat some of the material already discussed in this chapter of the report.

National CO Ambient Air Quality Standards

The National Air Quality Standards for carbon monoxide are 9 ppm (8 hour concentration) and 35 ppm (1 hour concentration). Both concentrations are, according to Figure 2, equivalent to 1.5 percent COHb, well below the minimum concensus level for safety. Doubling these standards to 18 ppm--8 hour concentration--and 70 ppm--1 hour concentration--would result in a COHb level of under 3 percent. This is slightly above the controversial value that Beard and Wertheim [2] reported was needed to impair task performance and below the value of 5 percent that according to general consensus should not be exceeded.

Knelson [11] states that "Data to place the national air quality standard for carbon monoxide on an irrefutable and unassailable scientific basis are simply not now available. The United States Environmental Protection Agency regulatory program. . . reflects the preventive medicine philosophy of maintaining a reasonable safety margin to protect all segments of the population from adverse health effects before they occur."

From the foregoing discussion it would seem that, in fact, a reasonably large safety factor has been included in the national CO ambient air quality standards.

3. Computing Emission Standards

Model Approach

It is generally agreed by both the automobile companies and the EPA that, of the models available today, the rollback or proportional technique is acceptable for relating air quality to automotive emission standards. It is understood that this type of model is a simplistic approach to a very complex atmospheric system. However, the more sophisticated diffusion models, which require vast data inputs of emission densities, traffic patterns, local meteorology, and topography, have not yet been developed to the point where they can provide better answers to the problem of specifically relating automobile emission standards to air quality standards.

The Proportional or Rollback Model

The proportional model assumes that local emissions and air quality are linearly related to one another and that growth in emission will produce a linear growth in atmospheric pollutant concentrations. By this reasoning, a reduction in emissions will result in a corresponding reduction in atmospheric pollutant concentrations.

The general form of the rollback model as derived by Larsen [12,13] and used by Barth [14] to compute 1980 automotive emission goals is

$$R = \frac{g(P) - D}{g(P) - B} \times 100, \qquad \text{Eq. 1}$$

where: R = % reduction in all sources necessary to meet air quality standards
D = air quality standards or goals in ppm or $\mu g/m^3$
P = present air quality in ppm or $\mu g/m^3$
g = growth factor
B = background concentration in ppm or $\mu g/m^3$

The term g(P), growth times present air quality, is equal to the future concentration of the pollutant if no controls are instituted. From the reduction percentage, R, the permissible automotive emissions can then be calculated. R applies to all sources. In the case of CO, emission inventory studies show that automobile exhausts are the primary sources of carbon monoxide in most urban atmospheres.

The percent reduction from Equation 1 is related to permissible automotive emissions by

$$E = A\left(1 - \frac{R}{100}\right), \qquad \text{Eq. 2}$$

where: E = permissible automotive emissions which will achieve the air quality standards
A = present automobile emissions
R = reduction percentage from Eq. 1

The automotive industry has offered another form of Equation 1, which computes the term $\left(1 - \frac{R}{100}\right)$ directly:

$$1 - \frac{R}{100} = \frac{(D-B)}{g(P-B)}. \qquad \text{Eq. 3}$$

Equations 1 and 3 are essentially the same and give almost the same results when used to solve for E in Equation 2.

Another approach to the rollback model is to use carboxyhemoglobin concentrations. Equation 1 then becomes

$$R = \frac{g(COHb_P) - (COHb_D)}{g(COHb_P) - (COHb_B)} \times 100, \qquad \text{Eq. 4}$$

where: $COHb_P$ = present carboxyhemoglobin concentrations as a result of present air quality
$COHb_D$ = carboxyhemoglobin concentration which corresponds to CO air quality standard
$COHb_B$ = background COHb found in normal individuals as a result of metabolic processes

Present Automobile Emissions (A in Equation 2)

As previously stated, the proportional rollback technique assumes that emissions and air quality are linearly related, and that reduction in emissions will result in a linear improvement in air quality. Thus, if we use 1970 air quality to compute an emission reduction, this reduction should be applied to 1970 emissions.

The 1970 emission baseline, as used here, means the average emissions for all cars driven during that year, not just the emissions from 1970-model cars; this is because 1970-model cars accounted for only 15 percent of the total miles driven in 1970. Thus, the 1970 emission baseline to be reduced must be the weighted emissions from all

cars. The baseline computed in this manner and converted to the currently required constant volume sample -- Cold/Hot start (CVS -- C/H) test protocol (Appendix B) is 71.2 g/mi CO.

4. Carbon Monoxide Air Quality

The Nature of Carbon Monoxide Data

Carbon monoxide air quality data in general appear to be log-normally distributed, i.e., the logarithms of CO concentration from a particular site over a long period of time show a Gaussian or normal distribution about the geometric mean concentration. When plotted on log-probability graph paper, this type of distribution produces a straight line. Carbon monoxide Continuous Air Monitoring Program (CAMP) data appear to exhibit this characteristic. Plots of 8-hour average Chicago and Philadelphia CO CAMP data [15] are given in Figure 4.

Urban carbon monoxide concentrations are not uniformly distributed throughout a city. As one would expect, high CO levels are found in the high traffic density area and low values in residential sections. Local meteorology and topography can greatly influence the ambient levels of CO. Diffusion models of canyon streets show that the concentrations can be 2 or 3 times higher on one side of the street than the other when the wind is at right angles to the street. Ambient measurements tend to verify this.

Selection of Value of Present Air Quality To Be Used in Rollback Computation (P in Equation 1)

Averaging Time and Frequency of Occurrence

For consistency, the values of P, D, and B in Equation 1 must have the same averaging time. The averaging times for values of D (air quality standards or goals) are stipulated by the National Air Quality Standards as 1-hour and 8-hour averaging times, respectively; and since there is not good argument for using other averaging times in Equation 1, the values of P (present air quality) and B (background concentration) should have the same averaging time as D, the National Air Quality Standards. Computations have shown that only the 8-hour average values need to be used to determine the value of R (percent reduction required).

The 8-hour average National Air Quality Standard for CO is 9 ppm, not to be exceeded more than once a year. However, this is

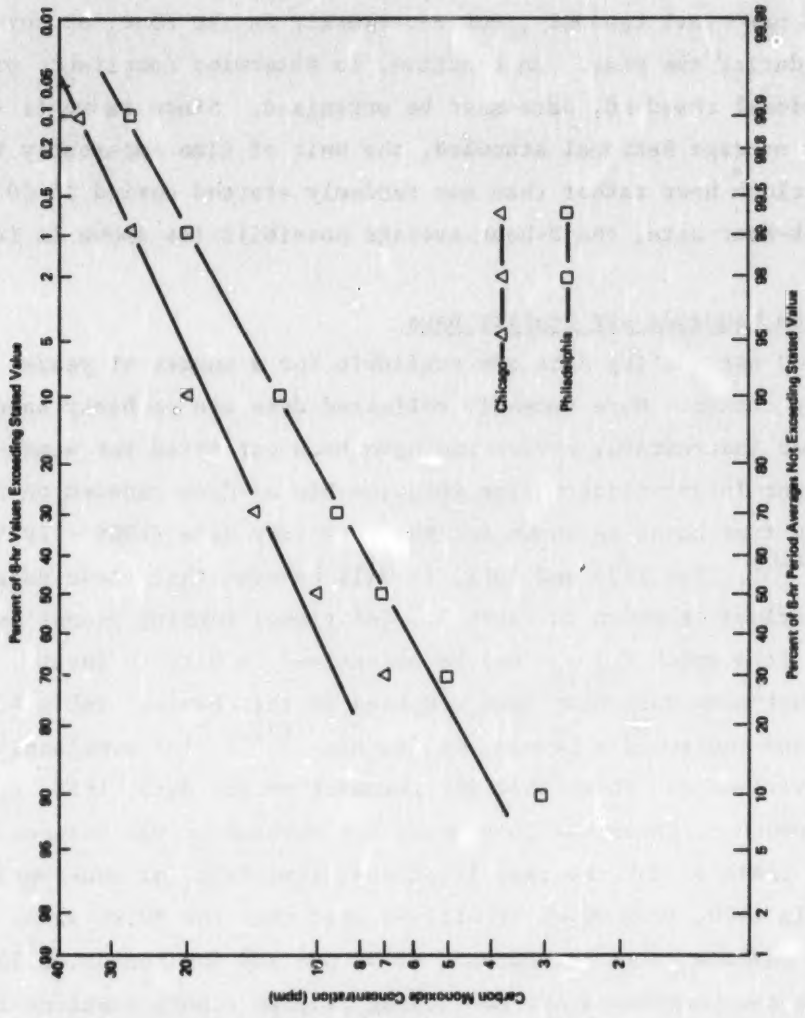

Percent of 8-hr Values Exceeding Stated Value
Percent of 8-hr Period Averages Not Exceeding Stated Value
Carbon Monoxide Concentration (ppm)
Chicago
Philadelphia

FIGURE 4 Concentration of carbon monoxide versus percentile [CAMP data, 1962 - 1968, 8-hr averaging time taken from Table 2 of Larsen (15)].

ambiguous since it does not specify which 8-hour period to use. There is an infinity of possible 8-hour running average values if the running averages may start randomly, not necessarily on the hour, at any instant of time during the year. In practice, to determine compliance with this National standard, data must be organized. Since there is also a one-hour average National standard, the unit of time apparently intended was the clock hour rather than any randomly started period of 60 minutes. For clock-hour data, the 8-hour average possibilities shown in Table 1 exist.

Carbon Monoxide Air Quality Data

CO air quality data are available for a number of years from many cities. More recently collected data are probably associated with fewer instrumental errors and have been corrected for atmospheric water vapor interferences. The relationship of data reduced on several averaging time bases is shown for New York City data (1969 - 1971) in Table 2 [16]. For 1970 and 1971, it will be seen that these data may be categorized as shown in Table 3. Additional running 8-hour average CO data, from which $P_{99.99}$* may be determined is hard to locate, because not many data have been computed on this basis. Table 4, for Burbank and Inglewood - Lennox, California [17] is for overlapping 8-hour averages and shows that for the most recent date, 1968, $P_{99.99}$ for Inglewood - Lennox was 30 ppm and for Burbank it was between 40 and 45 ppm. Table 5, for the same locations, give data for non-overlapping periods in 1970, from which it will be seen that the 99.9% value was less than the maximum value for Burbank of 32 ppm and for Lennox of 35 ppm. Data from the Continuous Air Monitoring Program (CAMP) stations in eight cities (Table 6) [15] are for calendar-day data so that both the $P_{99.9}$ value, based on all eight-hour periods or the $P_{99.7}$ value, based on only 8 a.m. - 4 p.m. periods for the most recent year of published data, 1968, must be less than 29 ppm for Denver, Philadelphia, St. Louis, and Washington, and less than 36 ppm for Cincinnati and Chicago. Figure 4, which is based on the same body of data for Chicago and Philadelphia as Table 6 shows that $P_{99.9}$ points for these cities for all

*The notation P_x means the present air quality concentration in ppm or g/m^3 not to be exceeded more than x percent of the time in a year (see Table 1 for example).

Table 1 Eight-Hour Averages Possible with Clock-Hour Data

Definition	Number of Possible 8-hr Periods/Yr		If One Period Exceeds Standard: % of Periods	
	Maximum	Minimum	Exceeding Standard	Not Exceeding Standard
Running 8-hr average	8760	8752	0.01	99.99
Three non-overlapping 8-hr periods per 24-hr period	1095	1094	0.1	99.9
Three non-overlapping 8-hr periods per calendar day[a]	1095		0.1	99.9
One specific 8-hr period per 24-hr period	365	364	0.3	99.7
One specific 8-hr period per calendar day	365		0.3	99.7

[a]These must be midnight - 8 a.m., 8 a.m. - 4 p.m., 4 p.m. - midnight

Table 2 8-Hour Maximum Carbon Monoxide Values from High Traffic Density Areas in New York City (ppm)[a]

Station	Running 8-hr Average		6 a.m. - 2 p.m.		7 a.m. - 3 p.m.		8 a.m. - 4 p.m.	
	Maximum	Second Highest	Maximum	Second Highest	Maximum	Second Highest	Maximum	Second Highest
Canal Street								
1969	26.4	26.1	25.8	22.8	26.4	23.4	26.1	22.8
1970	45.1	45.0	43.1	42.6	44.6	41.0	45.1	42.8
1971	51.9	50.5	41.5	35.6	36.1	35.5	37.1	35.6
59th St. Bridge								
1969	32.0	31.6	28.8	26.9	26.1	25.6	25.6	25.0
1970	45.7	44.6	43.1	42.5	42.4	40.8	40.3	40.1
1971	37.5	37.5	36.0	31.4	33.3	30.0	30.8	26.0
45th Street								
1969	58.4	56.4	58.4	47.3	56.4	50.4	54.9	50.5
1970	34.5	32.9	31.5	30.9	30.9	30.9	30.1	30.0
1971	29.3	29.0	27.5	26.5	27.5	27.4	27.1	26.9

[a]Computed at University of North Carolina from data made available by New York City.

Table 3 8-Hour Averages for Various Percentile Assumptions - High Traffic Density Areas in New York City

Definition[a]	Percentile -- P_x (x =)	CO Concentration -- ppm					
		1970			1971		
		59th Street	45th Street	Canal Street	59th Street	45th Street	Canal Street
Running 8-hr average	99.99	44.6	32.9	45.0	37.5	29.0	50.5
Three non-overlapping 8-hr periods per 24-hr period	99.9	42.5	30.9	42.8	31.4	27.4	35.6
Three non-overlapping 8-hr periods per calendar day	99.9	40.1	30.0	42.8	26.0	26.9	35.6
One specific 8-hr period per 24-hr period(e.g., 6 a.m. - 2 p.m.)	99.7	42.5	30.9	42.6	31.4	26.5	35.6
One specific 8-hr period per calendar day (e.g., 8 a.m. - 4 p.m.)	99.7	40.1	30.0	42.8	26.0	26.9	35.6

[a]See Table 1.

Table 4 Percent of All Overlapping 8-Hour Averages Having Values Equal To or Exceeding Specified Values (Concentration in ppm)[a]

Burbank	0	5	10	15	20	25	30	35	40	45
1963	100	94	30	8	3	2	1	0.4	0.1	0.05
1964	100	84	15	3	0.5	0.1				
1965	100	95	25	6	1.5	0.25	0.03			
1966	100	97	42	8	1.4	0.1				
1967	100	99	69	17	5	1	0.13			
1968	100	52	18	6	2	0.7	0.2	0.07	0.02	
Inglewood - Lennox[b]										
1964	100	96	48	11	3	1	0.1	0.05		
1965	100	99	88	37	14	5	1.35	0.25		
1966	100	90	46	16	4	1	0.13			
1967	100	70	18	6	1.7	0.48	0.12			
1968	100	44	13	4	1	0.3	0.01			

Source: Personal communication, John Maga to Richard Kamens[17].

[a] Water vapor interference (5 ppm) correction applied to data collected prior to 4/1/68.

[b] Station was relocated from Inglewood to Lennox in February 1965.

Table 5 8-Hour Non-overlapping Periods in 1970 with Average Concentration Values Equal to or Exceeding 9 ppm

Time and Location	No. of Periods	Max. 8-hr Avg. Conc.	Time and Location	No. of Periods	Max. 8-hr Avg. Conc.
January			July		
Burbank	44	31	Burbank	9	12
Lennox	40	21	Lennox	4	10
February			August		
Burbank	31	32	Burbank	20	14
Lennox	41	28	Lennox	12	16
March			September		
Burbank	17	20	Burbank	36	20
Lennox	32	17	Lennox	19	35
April			October		
Burbank	14	16	Burbank	31	26
Lennox	23	16	Lennox	25	16
May			November		
Burbank	11	11	Burbank	46	29
Lennox	16	27	Lennox	28	32
June			December		
Burbank	2	9	Burbank	46	28
Lennox	3	9	Lennox	54	21

Source: Personal communication, John Maga to Richard Kamens[17].

Table 6 8-Hour Averaging Time Data -- CAMP sites (ppm)
(8 a.m. - 4 p.m., 4 p.m. - 12 midnight, etc.)

	Maximum for Year						
	1962	1963	1964	1965	1966	1967	1968
Chicago	20	22	35	37	44	33	33
Cincinnati	--	--	19	21	12	17	36
Denver	--	--	--	27	29	30	29
Los Angeles	28	28	27	--	--	--	--
Philadelphia	36	35	27	26	34	23	26
St. Louis	--	--	18	18	18	14	12
San Francisco	--	18	14	--	--	--	--
Washington	19	34	18	17	22	23	21

Source: Larsen[15].

seven years' data (1962 - 1968) are 34 and 27 ppm respectively, although for individual years these points would be closer to the annual maximum values shown in Table 6. The 1970 maximum value for the Chicago CAMP station was 23 ppm, indicating a $P_{99.9}$ value of less than 23 ppm.

The NATO report [1] chapter on atmospheric carbon monoxide concentrations additionally reviewed data from Germany, England, France, and Italy and concluded that "An analysis of air monitoring data indicates that 0.1 percentile 8-hour-averaging-time urban CO concentrations vary from approximately 12 to 46 mg/m^3 (10 to 40 ppm)."

The value used by Barth [14] for his CO computation was the P_{100} value of 44 ppm for the Chicago CAMP station for 1966 (Table 6). In the array of data discussed above there is one station (45th St., New York City -- Table 2) that has higher values than this for one year (1969), but lower values in 1970 and 1971. The other two New York City stations exhibit slightly higher running averages but lower non-overlapping and specific-hour averages. The $P_{99.99}$, $P_{99.9}$, and $P_{99.7}$ averages from all other cities listed are less than 44 ppm, in fact, closer to 30 ppm. Therefore computations later in this report are based on a high value of 44 ppm and a low value of 30 ppm.

Selection of Value of Background Concentration To Be Used in Rollback Computation (B in Equation 1)

The nonurban global background concentration for carbon monoxide is about 0.1 ppm [18]. When compared to the standards the background concentrations are relatively low and have little influence on the computed reduction. There appears to be general agreement that an urban background value of 1 ppm be used in the rollback calculations.

5. Selection of Growth Factor To Be Used in Rollback Computations (g in Equation 1)

The original form of the rollback formula given by Larsen [12,13] simply relates air quality with air quality standards or goals:

$$R = \frac{P - D}{P} \times 100. \qquad \text{Eq. 5}$$

This permits the calculation of overall average air quality control that is necessary to meet air quality standards, if the present air quality is not degraded further by increasing emissions.

Present Federal requirements control only new automobiles. Hence the air quality standards, D, will not be achieved until all old automobiles without control devices are phased out. The year that air quality standards are expected to be met is called the "target year." A certain amount of increase in the number of automobiles, miles traveled, etc., must be expected, which will cause a rise in total emissions. In order to account for this, the present air quality, P, must be multiplied by a growth factor, g, during the period of concern (emission baseline year to target year) to yield the future air quality that would be expected at the target year if there were no controls placed on emissions. It is this future air quality that must then be reduced. Under these circumstances, Equation 5 becomes Equation 6:

$$R = \frac{g(P) - D}{g(P)} \times 100. \qquad \text{Eq. 6}$$

When background concentration is considered, Equation 6 converts to Equation 1.

Choosing a Growth Factor

Barth [14] and Romanovsky [19] used a growth factor based on the ratio of the number of motor vehicles expected in 1990 compared to those on the road in 1967. This resulted in a growth period of 23 years, a growth factor of 2.18 and a noncompounded growth rate of 5.1 percent. Barth assumed that the emission limits were to be imposed in 1980, and that 10 additional years were needed after 1980 to phase out current uncontrolled motor vehicles. Hence, the target date for attaining the air quality goals was estimated to be 1990. However, the Clean Air Act of 1970 imposed emission standards in 1975.

Following the above reasoning, the target year becomes 1985; the period of growth from a 1967 baseline becomes 18 years and, using the same growth rate of 5.1 percent, the growth factor becomes 1.92 for the period 1967 - 1985.

A different approach to estimating the growth period is to estimate future CO emissions (Figure 5) and to note that the 1975 standards will not cause a minimum emission to be reached until 1988. This yields a growth period from 1970 (with the enactment of the Clean Air Act of 1970) to 1988, also a period of 18 years.

More recently, Barth and Romanovsky [20] have cited other automotive related growth rates (Table 7).

Ott et al. [21] have calculated future carbon monoxide concentrations from emission density data by use of diffusion modeling techniques. The mean emission growth factor calculated by them for Washington, D.C., ranges from 1.32 at the center of the city to 2.46 at five miles from the city center for the period 1964 - 1985 (Figure 6). These growth factors correspond to a noncompounded growth rate of 1.15 percent and 6.9 percent, respectively. Within the city limits of Chicago, traffic density growth factors range from approximately 1 to 2.5 for 1956 - 1980 (Figure 7). It is generally agreed that high density traffic streets and arteries are essentially saturated now, and very little future increases in vehicle numbers can be expected near the center of large cities. Under these conditions, a growth factor based on the number of automobiles would be close to unity.

There is evidence [21] that a growth factor related to vehicle activity in the central city core could underestimate the concentration at a center city receptor site because carbon monoxide advection from upwind sources is not taken into account. Diffusion computations by Ott et al. [21] and Turner predict an increase in concentration at the city center due to emission growth outside of the city. Ott et al. found a concentration growth factor for 1964 - 1985 of from 1.44 - 1.69 for Washington, D.C. Fumarola (Appendix C), using a technique similar to that used by Ott et al., found a concentration related growth factor in the range of 1.40 - 1.57 for this same period. As shown earlier

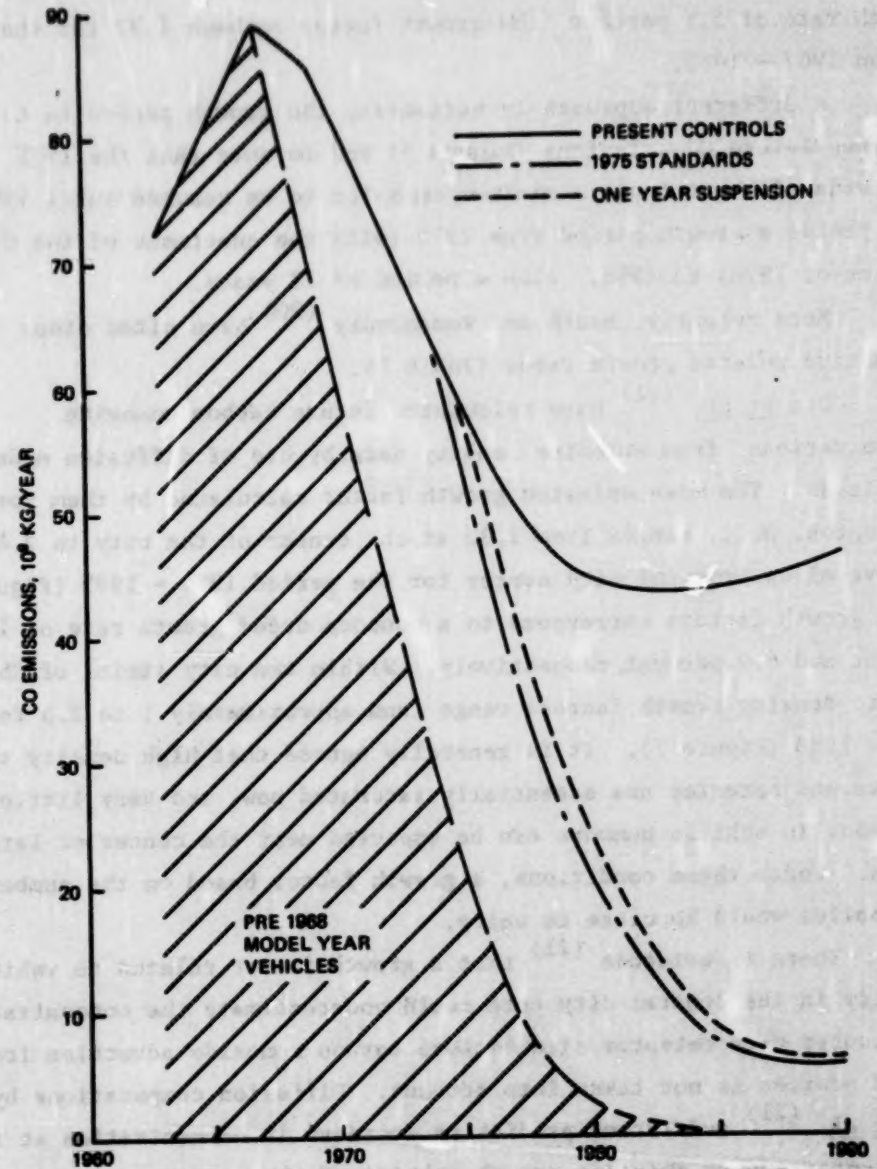
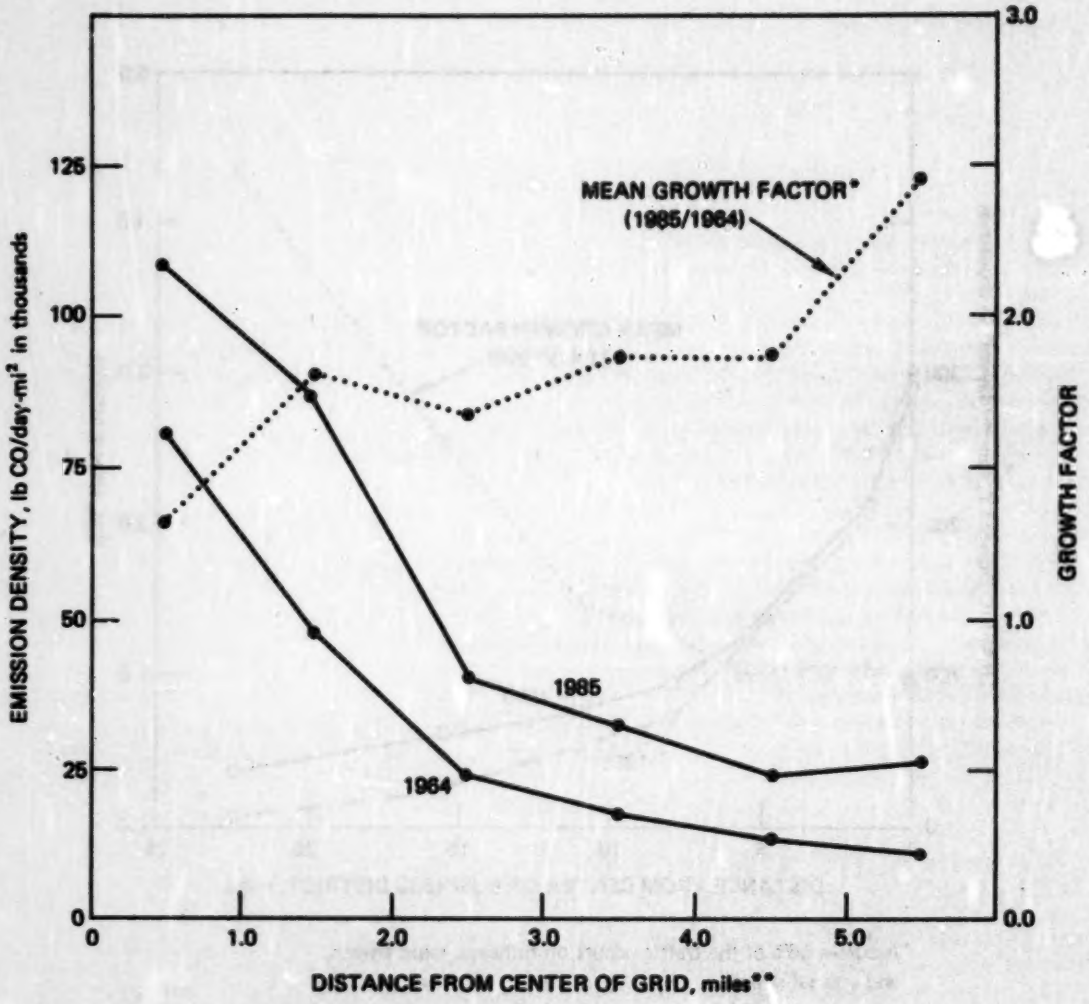
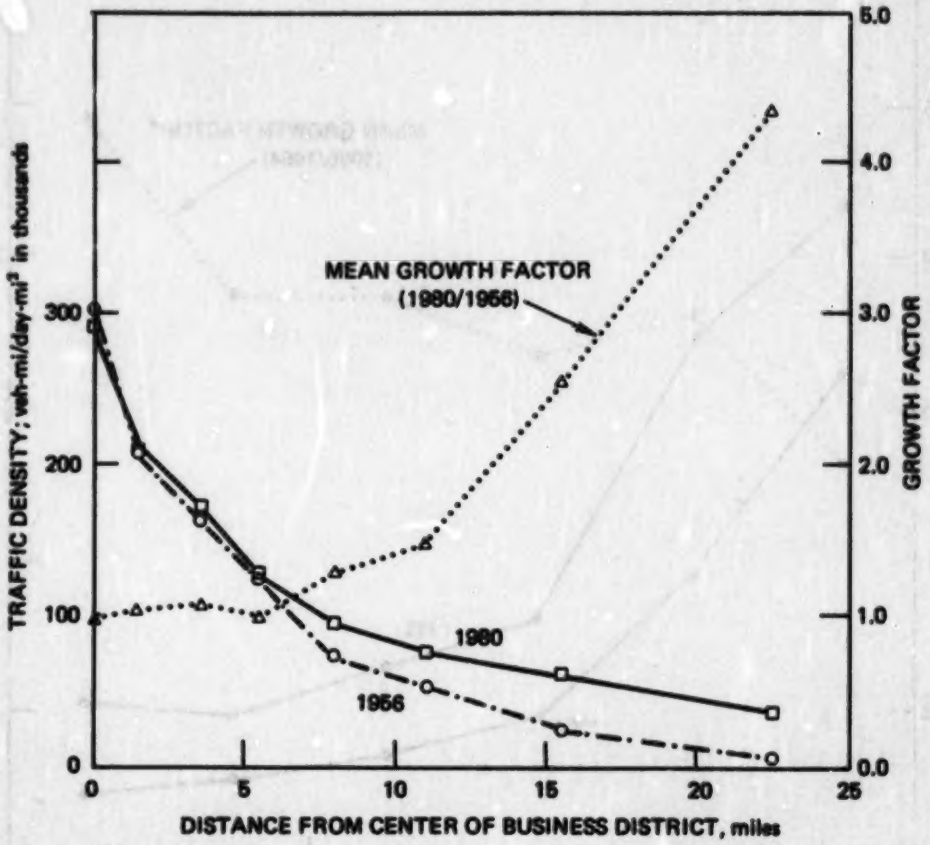


FIGURE 5 United States nationwide carbon monoxide emissions. Source: Semiannual report to Environmental Protection Agency, Committee on Motor Vehicle Emissions, National Academy of Sciences, Washington, D.C., p. 47, January 1, 1972.



*The average of all growth factors within a ring
**Distance is measured from the center of the grid to the midpoint of a ring.

FIGURE 6 Emission density profiles from Washington, D.C. Source: Ott et al. (21).



*Assumes 86% of the traffic occurs on tollways, expressways, and arterial streets.

FIGURE 7 Traffic density profiles for Chicago. Source: Ott *et al.* (21).

Table 7 Automotive Related Growth Rates

Parameter	Time Span	% Annual Growth
Average miles per vehicle	1959 - 69	0.26[a]
Motor Vehicle Registration	1959 - 70	4.79[a]
Motor Vehicle Registration	1960 - 85	3.7[a]
Gasoline consumption	1960 - 70	5.58[a]
Gasoline consumption	--	4.0
Gasoline consumption	--	3.2[a]
Total vehicle miles	1959 - 70	5.51[a]
Travel in Chicago	1953 - 59	3.5[a]
Traffic growth in Chicago central business district	1946 - 71	1.8[a]
Cars on Los Angeles - Santa Monica Freeway	1967 - 71	3.7
Manhattan Bridge and Tunnel traffic	1963 - 70	2.7
Traffic volume in St. Louis	1969 - 70	5.9 - 7.9[a]
Inbound traffic on Potomac River Bridge	--	5.0

Source: Romanovsky[19].

[a]Noncompounded.

in this section, 18 years is the period of growth for 1975 emission standards if a 1970 baseline is used. Normalizing the above growth factor ranges to an 18- instead of 21-year period of growth yields a concentration growth factor from 1.34 to 1.59 (1970 - 1988).

A suitable growth factor for proportional modeling lies somewhere between estimates based on increasing nationwide automobile registration and traffic activity in the downtown areas of the city. On this basis, a growth rate of 3 percent per year (noncompounded) is suggested for the periods 1967 - 1985 or 1970 - 1988; this yields a growth factor of 1.54 for these periods.

6. Rollback Calculations

Calculation of Reduction Percentage (R in Equations 1, 2, and 4)

From the foregoing discussion, it can be seen that there are several choices possible for values of D, P, g, and B in the calculation of R using Equation 1. The automobile manufacturers argue that separate factors of safety have been incorporated in the values for D, P, g, and B recommended by EPA for use in Equation 1 and that the multiplication of these factors of safety results in an ultimate factor of safety that is unnecessarily high and economically burdensome. If this overall factor of safety were to be reduced, it could be done by decreasing it for any one of, or a combination of, the factors in the rollback calculation.

D -- Desired air quality. If this value is chosen as the National Air Quality standard for Carbon Monoxide - 9 ppm for 8 hours, not to be exceeded more than once a year, the least controversy would be generated. This value is obviously acceptable to EPA, since they promulgated it. It incorporates a factor of safety. The automobile manufacturers suggest that if the factor of safety be decreased for the value of D, it could rise to 15 ppm and still be considered safe. Therefore, in the sample computations that conclude this section, values of D of both 9 and 15 ppm are used.

P -- Present Air Quality. EPA recommends a value of 44 ppm - this being the highest 8-hour value for 1966 in the Chicago CAMP station data. Arguments have been set forth earlier in this report to use it as the high value in these computations and to use 30 ppm as the low value. The value of 44 ppm contains a factor of safety with respect to 30 ppm. Therefore in the sample computations that conclude this section, values of P of both 44 and 30 ppm are used.

B -- Background Concentration. Since both EPA and the automobile manufacturers appear to recommend a value of 1 ppm for B, this is the only value used in the sample computations that conclude this report.

g -- Growth Factor. The original EPA recommendation for growth factor was for a time interval different from that enacted into the Clean Air Act of 1970. After adjustment of time interval to conform to that enacted, the value of g recommended by EPA is 1.92. Barth and

Romanovsky [20] have indicated that this could reasonably be relaxed to a value of 1.54. The automobile manufacturers argue for a value of 1.0. The discussion of growth factors earlier in this report indicates the reasonableness of values ranging from 1.34 to 1.54. The value of 1.92 is at the high end of the range of values. The value of 1.54 is more nearly the median of the set of numbers.

In the computations that conclude this section, values of g of 1.9, 1.5, 1.3, and 1.0 have been used to show the range of values of R, percent reduction, that result recognizing that the values of the growth factor are not accurate to the third significant figure.

R -- Percent Reducation Required. Table 8 contains the computations referred to above and shows a range of values of R from 90.3, when all EPA recommended values are used, to 51.7 percent, when all automobile manufacturers recommended values are used; with intermediate values when the factors of safety are relaxed from one or some combinations of the values used in the computation.

If one uses Equation 4 and a $COHb_P$ of 4 percent (COHb level found during inversion in Milwaukee), a $COHb_D$ value of 2 percent, a $COHb_B$ value of 0.5 percent and a growth factor of 1.5, the resulting required reduction is 72.7 percent.

In partial conclusion, therefore, the several combinations of values of D, P, B, and g that yield reduction values (R) of approximately 80 percent, and more specifically, the one marked with the asterisk (D = 9, P = 30, B = 1, g = 1.5, R = 81.8) appear to be reasonable and to still retain a sufficient overall factor of safety.

Calculation of Emission Standard (E in Equation 2)

As indicated in Chapter 3 the 1970 CO emission baseline (CVS -- H/C basis) is 71.2 g/mi. The reduction percentages from Table 8 were applied to this baseline (Table 9). The reduction of 81.8 percent noted in Chapter 6 yields a CO Emission Standard of 12.9 g/mi(CVS -- H/C start basis). These values do not include any factor to account for deterioration of performance of the control system over the lifetime of the vehicle.

If, instead of basing 1970 CO emissions on the mix of cars

Table 8 Rollback Reductions Computed from Different Combinations of P, D, B, and g

D -- desired air quality	9	9	9	9	9	9*	9	9	15	15	15	15	15	15	15	15
P -- present air quality in ppm	44	44	44	44	30	30	30	30	44	44	44	44	30	30	30	30
B -- CO background conc. in ppm	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
g -- growth factor	1.9	1.5	1.3	1.0	1.9	1.5	1.3	1.0	1.9	1.5	1.3	1.0	1.9	1.5	1.3	1.0
R -- reduction in percent	90.3	87.7	85.8	81.4	85.7	81.8	78.9	72.4	83.1	78.4	75.1	67.4	75.0	68.2	63.2	51.7

* Sample computation: $R = \frac{g(P) - D}{g(P) - B} \times 100 = \frac{1.5 \quad (30) - 9}{1.5 \quad (30) - 1} \times 100 = 81.8\%$

Note: Equation 4 yields R = 81.6%.

Table 9 Carbon Monoxide Emissions Standard (g/mi -- CVS Hot/Cold start basis) Resulting from Use of the Reductions Computed in Table 8

R -- % Reduction Required (from Table 8)	A -- Present Automobile Emissions 1970 Baseline (71.2 g/mi[a])
90.3	6.9
87.7	8.8
85.8	10.1
81.4	13.2
85.7	10.2
81.8[b]	12.9
78.9	15.0
72.4	19.6
83.1	12.1
78.1	15.3
75.1	17.7
67.4	23.2
75.0	17.8
68.2	22.7
63.2	26.2
51.7	34.4
1975 Federal regulations	3.4

[a] See Appendix B

[b] Sample Computation: $E = A\left(1 - \frac{R}{100}\right) = 71.2\left(1 - \frac{81.8}{100}\right) = 12.9.$

Note: R calculated from Equation 4 yields E = 13.1.

on the road as noted in Chapter 3 and discussed in detail in Appendix B, the computations of Table 9 had been based on the emissions from only 1970 vehicles, the values for A in Table 9 would have been lower by the ratio of the 1970 model emission (47 x 1.12 x 0.72 = 37.9 g/mi) to the average 1970 car population emission (71.2 g/mi). We believe, however, that an emissions baseline developed from the weighted emissions of all cars on the road in 1970 is the one to be rolled back, for it is the entire vehicle population, not just 1970 vehicles, that gave rise to high urban CO values in 1970.

In final conclusion, an emission limit for CO for 1975 approximately three times as high as that promulgated by EPA for 1975 vehicles (3.4 g/mi, CVS -- H/C start basis) would give assurance of not exceeding the 8-hour air quality standard of 9 ppm CO more than once per year.

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APPENDIX A Toxicological and Epidemiological Appraisals of Carbon Monoxide

Toxicological Appraisal of Carbon Monoxide -- Summary*

Carbon monoxide is absorbed exclusively via the lungs, and most of its toxic properties are a result of its reaction with hemoproteins. The primary effect of CO is mediated by its reaction with hemoglobin to form carboxyhemoglobin (COHb), thus reducing the oxygen-carrying capacity of the blood and accounting for about 80 percent of an inhaled dose of CO. The affinity of hemoglobin for CO is more than 200 times that for oxygen, indicating that COHb is more stable than oxyhemoglobin (O_2Hb). The presence of COHb also shifts the O_2Hb dissociation curve to the left, implying that, at the tissue level of the circulation, less oxygen is available to the cells because of the decrease in oxygen tension (P_{O_2}). About 20 percent of a given dose of CO passes from the intravascular to the extravascular pool, reacting primarily with myoglobin to form carboxymyoglobin (COMb). About 1 percent or less reacts with the heme-containing cytochromes, and, at CO concentrations commonly encountered in community air pollution, this reaction is unlikely to be of any physiological significance.

The most accurate analysis of relatively low blood COHb concentrations results from determination of the CO liberated from the blood when the hemoglobin is destroyed. The necessary equipment is quite costly, a tedious procedure is involved, and relatively large blood samples are required. Several other good methods exist for the determination of low blood CO levels. The normal "background" concentration of blood COHb is about 0.5 percent, and this is attributed to endogenous sources such as heme catabolism. The body uptake of exogenous CO is determined by the concentration, length of exposure, and ventilatory rate, as well as by many other factors. When the concentration remains constant, a state of equilibrium is reached

* Air quality criteria for carbon monoxide -- A report by the expert panel on air quality criteria, Committee on Challenges of Modern Society, North Atlantic Treaty Organization, U.S. Government Printing Office, Air Pollution Technical Information Center, Office of Air Programs, Environmental Protection Agency, Research Triangle Park, N.C., June 1972.

in which the partial pressure of CO (P_{CO}) in the pulmonary capillary blood is almost equal to that in the alveolar and ambient air. The rate of uptake of CO is fairly constant with respect to blood COHb until about one-third of the equilibrium value is reached, and then this uptake proceeds at a slower and slower pace. COHb concentration at equilibrium is about 0.16 times the exposure level of CO in ppm. A reliable mathematical model exists for predicting COHb levels under a variety of circumstances.

The observed toxic effects of CO in both animals and humans reflect the impairment of the oxygen transport system. The data on experimental exposures of animals and humans are shown in Tables A-1 and A-2.

Studies have shown that the long-term exposure of animals to CO may produce morphological changes in the heart and brain. Dogs exposed intermittently or continuously to 58 mg/m^3 (50 ppm) for 6 weeks developed abnormal EKG's after the third week. At autopsy, these same dogs showed mobilization of glia and dilatation of the lateral ventricles of the brain. Continuous exposure of dogs to 58 mg/m^3 for 3 months failed to show such changes, but significant increases in hemoglobin levels, hematocrits, and RBC counts were observed. Continuous and intermittent exposure of dogs to 115 mg/m^3 (100 ppm) for 6 weeks produced a dilatation of the chambers of the right heart and, occasionally, of the left heart, and some degeneration of heart muscle was also noted. Dogs exposed to 115 mg/m^3 for 5-3/4 hours a day, 6 days a week for 11 weeks developed a consistent disturbance of the postural reflexes and of gait, although no changes in the electroencephalogram (EEG) or the peripheral nerves were reported. At autopsy, there was some indication of cortical damage.

The effects of CO on the hematological system of animals suggest the possibility of adaptation to CO. Cholesterol-fed rabbits that were adapted (acclimatized) by exposure to 195 mg/m^3 (170 ppm) CO for 8 weeks showed an increase in the hemoglobin level during the first 5 weeks of exposure; the level stabilized during the following 3 weeks. When the CO concentration was increased to 405 mg/m^3 (350 ppm), a further increase in hemoglobin was observed.

In other studies, short-term exposure of animals to low levels of CO produced effects on the central nervous system. Rats exposed to 58 mg/m^3

Table A-1 SUMMARY OF EFFECTS OF CARBON MONOXIDE IN ANIMALS

Species	CO level	Length of exposure	COHb, %	Effect	Reference
Dogs	58 mg/m^3 (50 ppm)	6 hrs/day, 5 days/wk, for 6 wk 24 hr/day, 7 days/wk, for 6 wk	2.6 to 5.5	Brain: Mobilization of glia and dilatation of lateral ventricles. Necrosis and demyelination absent. Heart: 10/15 developed EKG changes in third week.	Lindenberg et al. (57)
Rats Rabbits Dogs	58 mg/m^3 (50 ppm)	24 hr/day, 7 days/wk, for 3 mo	Rats 1.8 Rabbits 3.2 Dogs 7.3	Dogs: No changes in EKG's and pulse rates. No histologic difference between exposed and control animals. Significant increases in hemoglobin levels, hematocrits, and RBC counts.	Musselman et al. (81)
Rats	58 mg/m^3 (50 ppm)	24 hr/day, 7 days/wk, for 3 mo		EKG changes in the first 2 weeks returning to normal in third week. Slight increase in hemoglobin levels, hematocrits, and RBC counts.	Roussel et al. (82)
Rats	58 mg/m^3 (50 ppm)	1 hr 1 to 5 hr/day for 4 days		Changes in EEG increasing in severity with length of exposure. Progressive deterioration in EEG returning to normal 48 hours after end of exposure.	Xintaras et al. (59)
Dogs	115 mg/m^3 (100 ppm)	6 hr/day, 5 days/wk, for 6 wk 24 hr/day, 7 days/wk, for 6 wk	7 to 12	Brain: Mobilization of glia and dilatation of lateral ventricles. Necrosis and demyelination absent. Heart: 8/8 developed abnormal EKG's after about 2 weeks. Autopsy: dilatation of right heart and occasionally of left heart; some degeneration of heart muscle.	Lindenberg et al. (57)
Dogs	115 mg/m^3 (100 ppm)	5-3/4 hr/day, 6 days/wk, for 11 wk	up to 21	Brain: No changes in EEG or in peripheral nerves. Consistent disturbance of postural reflexes and gait. Autopsy: 6/6 showed some indication of cortical damage. Heart: 1/4 had inverted T-wave after second week 2/4 had inverted T-wave by tenth week Autopsy: 4/4 showed degenerative changes in muscle fibers.	Lewey and Drabkin (56)

Table A-1 SUMMARY OF EFFECTS OF CARBON MONOXIDE IN ANIMALS

Species	CO level	Length of exposure	COHb, %	Effect	Reference
Rats	115 to 11,500 mg/m^3 (100 to 10,000 ppm)	up to 48 min.	n.a.	Impairment in time discrimination.	Beard and Wertheim (58)
Rabbits	195 mg/m^3 (170 ppm)	8 weeks	19.7 at end of first week	Decrease in % COHb from 19.7 to 15.1. Increase in hemoglobin level in first five weeks followed by stabilization. Exposure to 400 mg/m^3 (350 ppm) caused further increase in Hb. Rats were cholesterol fed. Increased total cholesterol in aortic tissue in rabbits exposed to CO.	Astrup et al. (86)
Dogs	920 to 1150 mg/m^3 (800 to 1000 ppm)	6 to 8 hr/day, 7 days/wk, for 36 wks		Increased RBC count, hemoglobin, and blood volume. Challenge with 575 mg/m^3 (500 ppm) in these dogs produced the same % COHb as in non-acclimatized controls.	Wilks et al. (124)
Dogs	>1,150 mg/m^3 (1,000 ppm)	n.a.	3 to 40	Prominent EKG changes. High mortality rate. Alterations in cerebral and myocardial histology.	Prezioso (83)
Dogs	115 mg/m^3 (100 ppm)	6 wk, continuous and intermittent	7 to 12	EKG changes, more marked with continuous than intermittent exposure. Dilatation of right cardiac chamber. Dilatation of lateral ventricles of the brain.	Prezioso (83)
	58 mg/m^3 (50 ppm)	6 wks, continuous and intermittent	2.6 to 5.5	Same changes as noted with exposure to 115 mg/m^3, except EKG changes were not as prominent.	
Mice	58 mg/m^3 (50 ppm)	3 mo to 2 yr	n.a.	No changes noted in fertility, fetal survival, body growth, food intake, weight and water content of various organs, EKG, or blood chemistries.	Stupfel (61)
Monkeys	55 mg/m^3 (48 ppm)	n.a.	3.7 to 4.7	No decrement in performance, even at simulated altitude of 27,000 feet.	Back and Dominguez (62)

Table A-2 Summary of Effects of Carbon Monoxide in Humans

No. Subjects	CO level	Length of exposure	COHb, %	Effect	Reference
7	115 to 23,000 mg/m^3 (100 to 20,000 ppm)	Up to 5 hr	Up to 35.0	Uptake of CO increases with concentration of CO, length of exposure, and ventilation rate. Rate of uptake of CO as measured by increase in blood % COHb is constant up to 1/3 of equilibrium COHb. Rate of uptake decreases with increased P_{O_2} and apparently increases with decreased P_{O_2} due to hyperventilation. When the latter is corrected for, rate of uptake is unaltered.	Forbes et al. (A-12)
10	35 mg/m^3 (30 ppm)	24 hr	5	Approximate equilibrium value of COHb reached by 12 hours; 60% of equilibrium value reached in 2 hours, 80% in 4 hours, and the remainder over 8 hours.	Smith (A-13)
4	100% (100 to 300 ml)	10 to 15 min	Up to 20	Impairment of visual function detectable at approximately 4.5% COHb and increases with increase in COHb. Recovery lags behind excretion of CO. Latter depends on composition of gas during post-exposure period. Experiments at simulated altitudes gave a similar pattern of results. Data given for one subject only.	Halperin et al. (A-14)
1	n.a. (1 cigarette)	n.a.	2	Impairment of visual function.	MacFarland et al. (A-15)
1	n.a. (3 cigarettes)	n.a	4	Impairment of visual function similar to that produced by an altitude of 8000 ft.	
49	n.a.	n.a.	Up to 20.4	Impairment in response to certain psychomotor tests, detectable at 5% COHb. No effect on pulse, respiratory rate, blood pressure, neurological reflexes, muscle persistence, or static steadiness test	Schulte (A-16)

Table A-2 Summary of Effects of Carbon Monoxide in Humans

No. Subjects	CO level	Length of exposure	COHb, %	Effect	Reference
18	58 to 230 mg/m^3 (50 to 250 ppm)	Up to 2 hr	2.5	Significant impairment in time-interval discrimination after exposure to 58 mg/m^3 (50 ppm) for 90 minutes	Beard and Wertheim (A-6)
4	58 to 230 mg/m^3 (50 to 250 ppm)	Up to 2 hr	3	Consistent impairment in 3 or 4 parameters of visual function after exposure to 58 mg/m^3 for 50 minutes.	Beard and Grandstaff (A-17)
18	29 to 1,150 mg/m^3 (25 to 1000 ppm)	Up to 16 hr	Up to 31.8	No impairment of time estimation. No change in blood chemistry.	Stewart et al. (A-18)
	Up to 115 mg/m^3 (100 ppm)	8 hr	11 to 13	No impairment in psychomotor test performance.	
42	115 mg/m^3 (100 ppm)	3 ½ hr	7.2	Impairment of visual perception, of manual dexterity, of learning, and impairment of performance of certain intellectual tasks.	Bender et al. (A-19)
20	58, 115, and 174 mg/m^3 (50, 100, and 150 ppm)	110 min	not measured	Impairment of auditory vigilance: percentage of missed signals is correlated to CO concentration in inspired air. Impairment of EEG.	Groll-Knapp et al. (A-20)
12	58 mg/m^3 (50 ppm)	5 hr	not measured	Impairment of auditory vigilance between the 80^{th} and 125^{th} min of exposure. No changes in psychomotor tests.	Fodor and Winneke (A-21)
5	n.a.	Up to 2 min	Above 5	Decreased P_{aO_2} and P_{vO_2}.	Ayres et al. (A-22)
26	n.a	Up to 2 min	Above 5 (average 8.96)	Increased oxygen extraction in the presence of COHb.	Ayres et al. (A-23)
111	n.a	Up to 2 min	Above 5 (average 8.5 to 9)	Increased lactate production in patients with coronary heart disease; increased coronary blood flow, and fall in mixed venous and coronary sinus P_{O_2}.	

(50 ppm) for 1 hour developed alterations in the electroencephalogram (EEG), which increased in severity with longer periods of exposure. These changes returned to normal 48 hours after the end of the exposure. After exposure to 58 mg/m^3 for 48 minutes, rats developed an impairment in time discrimination. Short-term exposure to higher CO concentrations produced similar effects, but with increased severity.

No long-term human studies on experimental CO exposures have been reported, although there are data on occupational exposures. Brief exposures to high levels of CO produced effects on the central nervous, vascular, and respiratory systems. Central nervous system effects apparently occurred at COHb levels above 2 percent. Exposure of nonsmokers to 58 to 290 mg/m^3 (50 to 250 ppm) CO for up to 2 hours produced a significant impairment in time discrimination, statistically significant at about 58 mg/m^3 for 90 minutes (approximately 2.5 percent COHb). At blood COHb levels of about 3 percent (estimated by expired air analysis after exposure to 58 mg/m^3 of CO for 50 minutes), significant changes in relative brightness threshold and visual acuity were observed. Impairment in the performance of certain psychomotor tests occurred at about 5 percent COHb. Exposure of four subjects to 100 to 300 ml of pure CO for several minutes produced an impairment in visual function, which was significant when the blood COHb level in one subject reached about 4.5 percent.

Exposure to CO affects oxidative metabolism of both systemic and myocardial circulatory beds. Blood COHb levels above 5 percent in five subjects produced a mean decrease of 13.3 percent in mixed venous oxygen tension and 7.3 percent in arterial oxygen tension. In 26 subjects whose mean COHb was 8.9 percent, there was a mean decrease in the venous P_{O_2} that could be correlated with an increase in COHb. There were also associated increases in oxygen extraction ratios, ventilation, and cardiac output.

Myocardial metabolic studies have demonstrated that concentration of CO sufficient to produce significant increases in CCHb may produce a severe burden in subjects who already have increased demands on their oxygen supply, such as persons with coronary artery disease. Eleven cardiac patients, four of whom had coronary artery disease, were exposed

to CO that produced a mean COHb level of almost 9 percent, with several patients having blood COHb levels in the range of 5.5 to 6.5 percent. Patients with noncoronary heart disease showed a compensatory increase in coronary blood flow in response to the increased myocardial oxygen needs; this response did not occur in the four patients with coronary heart disease. In addition, the inadequate oxygen supply to the myocardium was reflected in the lactate extraction ratio, which changed to production in these four patients. It thus appears that people with certain forms of heart disease may be particularly susceptible to exposures of CO that could lead to blood COHb levels in excess of 5 percent. Many people with preclinical coronary artery disease may also have diminished myocardial performance at these levels.

The effects of CO are especially important at high altitude because of the lowered P_{O_2}. Both physiological and psychomotor tests have been employed to demonstrate these effects. As indicated by the increase in exercise pulse rate, each increase of blood COHb of 1 percent, up to 13 percent, has been shown to be equal in effect to about 100 m (335 ft) of altitude, applied over the altitude range of 2130 to 3050 m (7,000 to 10,000 ft). Impairment of visual threshold perception in a single subject at 2130 m (7000 ft) was equivalent to that produced by 5 percent COHb at sea level. In five subjects, a COHb level of 8 to 10 percent has produced a mean impairment in flicker fusion frequency (FFF) that was equivalent to lowering the altitude tolerance 1220 m (4000 ft) from about 3050 to 1830 m (10,000 to 6,000 ft).

In many of the human studies, the brief exposures to very high levels of CO make it difficult to relate the observed effects to equilibrium COHb levels. For long-term exposure to CO, certain effects such as increased hematocrits, hemoglobin levels, and blood volume may be present, but the available data are inadequate for drawing firm conclusions concerning the significance of all of these changes.

In summary, appraisal of short-term experimental exposures of humans to CO shows the following results in terms of the blood COHb levels observed:

1. No human health effects have been demonstrated nor have they been observed for COHb levels of 0 to 1 percent, since endogenous CO production makes this a physiological range.

2. The following effects on the central nervous system have been observed at 2 to 5 percent COHb:

a. At an estimated level of about 2.5 percent COHb in nonsmokers (based on exposure to 58 mg/m^3 CO for 90 minutes), an impairment in time-interval discrimination has been demonstrated.

b. At COHb levels of about 3 percent (based on exposure to 58 mg/m^3 for 50 minutes), changes in visual acuity and relative brightness threshold have been documented.

c. At about 5 percent COHb, an impairment in the performance of certain other psychomotor tests, and an impairment in visual discrimination, have been demonstrated.

3. The following cardiovascular changes have been observed at COHb levels of 5 to 10 percent:

a. In patients without coronary heart disease, changes were noted in cardiac output (increased), systemic arterio-venous oxygen-content difference, systemic oxygen extraction ratios, myocardial arterio-venous oxygen-content difference, and coronary blood flow.

b. In patients with coronary heart disease, lactate extraction changes to production and the compensatory increase in coronary blood flow is absent.

c. In patients with preclinical coronary artery disease, abnormal EKG's may increase.

Long-term experimental exposure of humans to CO may produce certain adaptive effects such as increased hemoglobin and hematocrits, but the available data are inadequate for drawing firm conclusions. Such effects have been observed in animals. There is a definite need to evaluate further the effects of cigarette smoking on the central nervous and cardiovascular systems, and to evaluate as well the possible "adaptation" of the cigarette smoker to CO.

Epidemiological Appraisal of Carbon Monoxide -- Summary*

Exposures to environmental levels of CO can increase blood COHb concentrations in human subjects. The amount of this increase is reasonably predictable and must be considered in relation to CO exposure from inhaled cigarette smoke and from occupational and domestic sources. Methods for estimating COHb levels in large populations are relatively simple.

Continuous exposure to relatively low levels of CO may result in significant increases in blood COHb levels over exposure periods of only a few hours. Such an exposure increases the body burden of COHb in persons who do not already have such a body burden from cigarette smoking. Longer exposures would most likely produce a somewhat greater increase, with greater potential for impairment of tissue oxygenation.

Exposure of traffic policemen, in Paris, for 5 hours to 12 to 14 mg/m^3 (10 and 12 ppm) increased COHb levels in nonsmokers by about 0.7 percent. The same exposures of cigarette smokers may cause those who have elevated COHb at the start of exposure to have a decrease in COHb and those who had relatively low values of COHb at the start of the study to have an increase in COHb. Cigarette smoking was not permitted during this 5-hour period of exposure.

Apart from increases in COHb, four possible effects have been a source of major consideration in epidemiologic studies:

1. Continued exposure to low levels of CO may produce some persistent toxic reaction, such as a chronic CO poisoning syndrome. The evidence for the occurrence of such a condition is inadequate and is based primarily on subjective symptoms.

2. Data suggest that weekly average CO values in excess of 9 to 16 mg/m^3 (8 to 14 ppm) may be associated with an increase in mortality in hospitalized patients with myocardial infarction.

* Air quality criteria for carbon monoxide -- A report by the expert panel on air quality criteria, Committee on Challenges of Modern Society, North Atlantic Treaty Organization, U.S. Government Printing Office, Air Pollution Technical Information Center, Office of Air Programs, Environmental Protection Agency, Research Triangle Park, N.C., June 1972

Substantiation of this impression will require a study of the prognosis of myocardial infarction patients in relationship to COHb levels measured at admission to the hospital.

3. It appears that drivers in commuting traffic undergo temporary reduction in blood O_2 content. Whether this amount of O_2 reduction results in refined, subtle impairment of the central nervous system is difficult to conclude, but there is suggestion that in some cases it may. It appears that driving in commuting traffic ordinarily offers very little health hazard or accident risk to a young, healthy, nonsmoking driver. This may not be so for older, hypoxic drivers. In one study, 30 such drivers after 90 minutes in the same traffic had 3.06 volume-percent less O_2 in capillary blood and had 28.3 percent slower reaction time than 30 young, healthy drivers; thus, older, hypoxic drivers may be potential accident risks. The difference of 100 msec in reaction time may make the difference between safety and mishap. When smoking is considered, such conclusions are further strengthened.

4. In two studies, persons driving motor vehicles that were involved in accidents had higher COHb levels than "control" populations; controls were not ideal, however. Possible mechanisms by which CO might affect the ability to drive a motor vehicle are suggested in the available data on CO effects upon visual sensitivity, psychological test performance, and accurate estimation of time intervals. As little as 2.5 percent COHb has produced these effects in laboratory studies of nonsmokers, and the available epidemiologic information is consistent with the premise that such an increase in COHb levels among drivers might influence the frequency of accidents.*

* A concluding section stating "Specific areas in which research is needed . . ." has been omitted from this summary.

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APPENDIX B Computing Total Carbon Monoxide Emissions from Light-Duty Vehicles

To arrive at the total CO emissions from light-duty vehichles during a given year, a weighted emission average is computed from all vehicles driven during that year. The weighted average is based on:

1. The emissions for each model year vehicle (Table B-1)
2. A deterioration factor to account for increased emissions as the motor vehicle becomes older (Table B-2)
3. Percentage of travel versus age of car (Table B-3)

The product of the emission rate, deterioration factor and percentage of miles traveled is found for each model year car driven during the year of concern. The sum of these products is total weighted emissions for the given year. An example calculation is shown in Table B-4.

The 1967 baseline of 112.67 given by Romanovsky* is a composite total emissions value for all motor vehicles during 1967. In a letter to Donald Schwentker of the Automobile Importers of America from Eric Stork of EPA, a baseline of 125 g/mi (c.v.s. cold start) is given along with the average emission rate for "well tuned" 1966 and 1967 cars (84.4 g/mi). In our calculations, we employed the 84.4 g/mi for uncontrolled 1967 model year cars as given by Stork and applied the deterioration and mileage factors as shown in Table B-4. This method produced a 1967 emissions baseline of 109.4 g/mi. To avoid confusion, we have elected to use the Romanovsky 1967 baseline of 112.67 g/mi. For 1968 - 1970 we used the baselines derived from the procedure given in Table B-4. The resulting baselines are listed in Table B-5.

*Romanovsky, J. C., Proportional model for relating motor vehicle emission requirements to air quality goals, Transcript of report presented before the Committee on Motor Vehicle Emissions of the National Academy of Sciences, Washington, D. C., pp. 1 - 13, February 10, 1972.

Table B-1 Light-Duty Vehicles CO Emission Rates by Model Year (Based on Well-Tuned Engines)

Year	Emission Rate (g/mi)[a]	References
Up to 1967	84.4	Letter from Eric Stork to Donald Schwentker
1968	72	Summary of Federal Motor Vehicle Emission Standards (LDV) EPA
1969	72	
1970 - 1971	47	1970 CO Emission Standards
1972 - 1974	39	1972 - 1974 CO Emission Standards
1975	4.7	1975 CO Emission Standards

[a] All emission rates are in terms of cold start CVS.

Table B-2 Deterioration Factor versus Age

Age of Vehicle (yr)	Deterioration Factor[a]
0 - 1	1.12
1 - 2	1.22
2 - 3	1.26
3 - 4	1.29
4 - 5	1.34
5 and over	1.40[b]

[a] These values were developed from EPA deterioration factor regression equations and are somewhat higher than those based on California Air Resources Board field surveillance data.

[b] The total CO emissions were not greatly affected when a deterioration factor in the range of 1.35 - 1.44 was used for cars five years old and older. Hence, a deterioration factor of 1.40 was arbitrarily selected for this group of cars to adjust the emission rates shown in Table B-1.

Table B-3 Vehicle Travel versus Age

Age (yr)	Travel Contribution	Total Miles Travelled
0	15.74	13,200
1	13.69	25,200
2	12.02	36,200
3	10.04	45,800
4	9.36	55,200
5	8.18	63,900
6	7.55	72,500
7	6.52	80,600
8	5.24	87,900
9	4.31	94,900
10	2.83	100,600
11	1.74	105,500
12 and older	2.78	109,800

Source: Bostich, T. A., and Greehalgh, H. J., Relationship of Passenger Car Age and Other Factors to Miles Driven, Washington, D. C.: U.S. Department of Commerce, Bureau of Public Roads, January 1967.

Table B-4 Sample Calculation of Total Emissions in g/mi from Light-Duty Vehicles during 1970 (Cold Start -- CVS Basis)

Year	Column 1 Emission Rate from Table B-1	Column 2 Deterioration Factor from Table B-2	Column 3 Fraction of Miles Traveled, Table B-3	Product of Columns 1 - 3
1970	47.0	1.12	.157	8.264
1969	72.0	1.22	.137	12.034
1968	72.0	1.26	.120	10.887
1967	84.4	1.29	.100	10.886
1966	84.4	1.34	.094	10.631
1965	84.4	1.40	.082	9.689
1964	84.4	1.40	.076	8.980
1963	84.4	1.40	.065	7.680
1962	84.4	1.40	.052	6.144
1961	84.4	1.40	.043	5.080
1960	84.4	1.40	.028	3.308
1959	84.4	1.40	.017	2.009
1958	84.4	1.40	.028	3.308
		Total Emissions from 1970		98.9
		Conversion factor to CVS/H/C basis		0.72
				71.2 g/m
				CVS -- H/C basis

Table B-5 Carbon Monoxide Emission Baselines

Year	Baseline Emissions of CO in g/mi Cold Start -- CVS	CVS -- Hot/Cold Start
1967	112.7	81.1
1968	107.7	77.6
1969	105.3	73.8
1970	98.9	71.2

APPENDIX C Computation of Concentration Growth Factors*

In order to evaluate a ground level concentration at any distance from a uniform area source a mathematical model was developed by Ott et al.** For our purposes this model has been modified as follows to apply to nonuniform area sources in a city (Equation C-1 and Figure C-1).

$$\chi = \int_0^{y-\epsilon} \frac{2.3\ q(x)}{u^{0.55}\ (y-x)^{0.75}}\ dx, \qquad \text{Eq. C-1)}$$

where: χ = pollutant concentration (grams per cubic meter)

$q(x)$ = emission density function of the distance from origin (grams per second per square meter)

u = wind speed (meters per second)

x = distance of the source from origin (meters)

y = distance of the receptor point from origin (meters)

ϵ = an infinitesimal positive number for numerical integration

What this model does is to calculate the concentration at receptor point, A, as the sum of all the infinitesimal sources upwind of the receptor. By moving receptor point, A, from the center downwind to the periphery of the city, a concentration profile is formed.

This model has been applied to Washington, D.C., for the years 1964 and 1985 (Figure 6 of this report). An equation was fitted to the emission density curves of Figure 6. The emission densities from these curves were then substituted for $q(x)$ in Equation C-1 to compute values of χ. 1964 and 1985 profiles for χ were computed for wind speeds of 2 to 12 meters/sec. For each wind speed, the ratios of 1985 to 1964 concentrations (χ) were computed and were called "concentration growth factors." Almost no influence of wind speed on concentration growth factor was noted. The concentration growth factor varied from 1.40 to 1.57 (Figure C-2).

The ratios of the 1985 to 1964 emission density values obtained from the above-noted fitted curves were called "emission growth factors" (Figure C-2). It will be seen from Figure C-2 that the concentration growth

* This appendix was prepared by Guiseppe Fumarola, Faculty of Engineering, University of Genoa, Italy.

** Ott, W., Clark, J., and Ozolins, G., Calculating future carbon monoxide emissions and concentrations from urban traffic data, U.S. Department of HEW, Public Health Service, Bureau of Disease Prevention and Environmental Control, Public Health Service Publication No. 999-AP-41, Cincinnati, pp. 11 - 21, 1967.

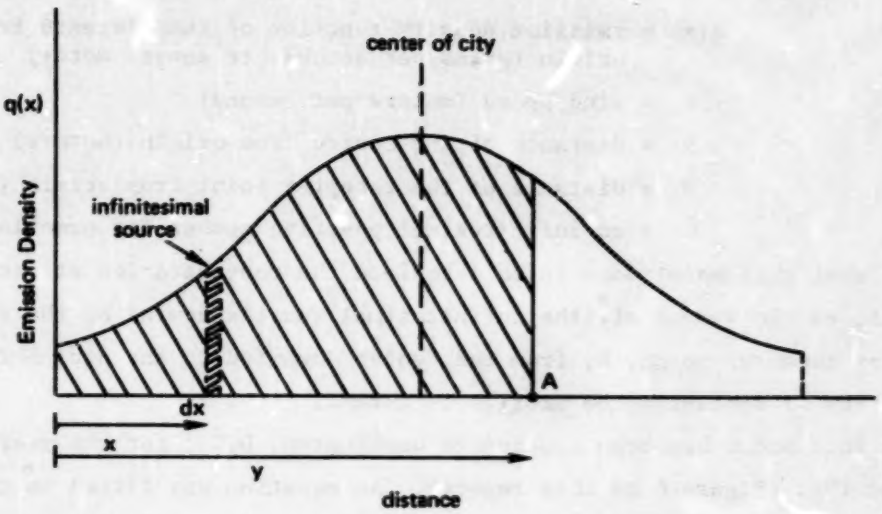

FIGURE C-1 distribution of emission density in the city.

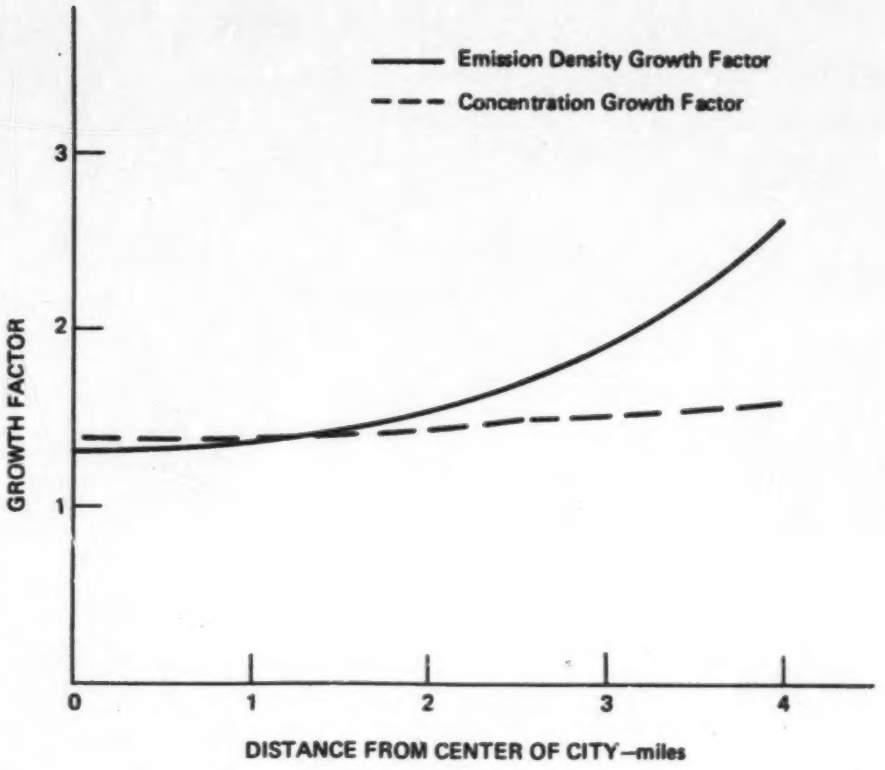

FIGURE C-2 Emission and concentration growth factor for Washington, D.C. (1964 - 1985).

factor is less influenced by distance form the center of the city than in the emission growth factor. It would appear that concentration growth factors computed in this manner are more meaningful values of g in the rollback reduction formula than are emission growth factors, particularly since P, D, and B are all expressed as concentrations.